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Highlights of this issue

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Assertive community treatment, ethnicity research and conceptual models

This issue of the *Journal* has a somewhat iconoclastic flavour, commenting on the lack of any clear benefit in outcomes of assertive community treatments over 'treatment as usual'; the problems of reporting results contrary to perceived wisdom in the case of ethnic differences in incidence of psychosis; and challenges, and alternatives, to the predominant biopsychosocial model in psychiatric practice. Killaspy *et al* (pp. 81–82) report that assertive community treatment did not reduce the need for in-patient care and that community mental health teams were able to prevent admissions equally successfully. These results were apparent at both the 18-month and 3-year follow-ups. An accompanying reappraisal piece, by Burns (pp. 5–6), suggests that ignoring the 'treatment as usual' arm of comparative studies has contributed to a delay in accepting the conclusions of this and similar studies. Instead of being viewed as an active comparator, this treatment arm was treated as a placebo; this led to a failure to support the utility of community mental health teams, which in simple terms yielded the same results at half the cost. He suggests that the removal of the term 'treatment as usual' from similar community psychiatry research would benefit both research and policy. An editorial argues that it is difficult to unshackle science from politics, and highlights the issues raised following the publication of a report arguing that ethnic differences in incidence and care pathways in psychosis were not due to institutional racism. Singh (pp. 1–2) makes a call for supporting academic freedom to ensure that the best evidence is available to inform practice, and that this forms a necessary counterbalance to combat prevailing attitudes and political expediency. Ghaemi (pp. 3–4) argues that the biopsychosocial model in psychiatric practice was useful in the past to counter an overly reductionist medical model of illness, but as the complexity of mental illness becomes better accepted, there are alternative models, which he reviews in his editorial, that may serve psychiatric practice better.

Depression: genes, environment and therapy

There has been an explosion of research interest in gene–environment interactions, and Goodyer *et al* (pp. 39–45) examined genotype, cortisol and development of depression in adolescents at risk for depression. This adds a physiological measure to extend earlier work showing that social adversity increases the liability to develop depression, through the presence of a short allele within the serotonin transporter gene. They found that the short allele of the serotonin transporter gene promoter was associated with higher morning cortisol levels and the subsequent development of a depressive episode. They conclude that these results may provide the mediating link with the hypothalamic–pituitary–adrenal axis necessary for the development of depression. Migration is a major environmental change and as most migrants are young adults, they tend to leave older family members behind, which is likely to have a detrimental effect on the latter's mental health. Abas *et al* (pp. 54–60) tested the intuitive hypothesis that these ageing parents will have higher rates of depression following such loss of close contacts. They collected data from Thailand and found that the hypothesis was not supported, and when relevant covariate factors were considered, there was actually less parental depression in those parents whose children had migrated. They speculate that this could arise through two mechanisms, either prior advantage in the migration group, or subsequent economic benefit secondary to money sent back to the parents. Cognitive–behavioural therapy (CBT) is an effective treatment for depression, and it is now possible to deliver this via computer; this is an efficient and cost-effective method – if it works. In a community sample of individuals with mild to moderate depression, de Graaf *et al* (pp. 73–80) found that unsupported computerised CBT did not do better than the routine treatment group. They conclude that this is not a useful intervention for this group and that it may be necessary to supervise this approach using a therapist, or to use it only as part of a stepped-care approach at a later stage of illness.

Diagnostic groupings, personality disorder and bipolar disorder

The prevalence and correlates of personality disorder across different countries remain unclear. Huang *et al* (pp. 46–53) used data from the 13-country World Health Organization world mental health initiative and reported prevalence rates of 6% for any personality disorder. They found increased rates of cluster A in males, a higher prevalence of cluster A and B personality disorders in the young and poorly educated. Personality disorders were associated with significant functional impairment and a high level of comorbidity with Axis 1 disorders. There was cross-national variation, with rates highest in Colombia and the USA and lowest in Western Europe and Nigeria. Bipolar disorder can be subclassified using various diagnostic criteria, including the DSM and the Research Diagnostic Critera (RCD). Hamshere *et al* (pp. 23–29) used data from the large Wellcome Trust Case Control Consortium to test the relative genetic support for these different operational diagnostic categories, and found that the RDC schizoaffective disorder, bipolar type, appeared to define a genetically homogeneous group, or one that had a strong genetic contribution. They suggest that this may have clinical importance and represents evidence for retaining this particular grouping in any future versions of DSM or ICD diagnostic schemes.